SAVING EYESIGHT AFTER MID-LIFE

WAITE

HARVARD HEALTH TALKS

The Care of Children. By John Lovett Morse.

Preservatives and other Chemicals in Foods. Their Use and Abuse. By Otto Folin.

The Care of the Skin. By Charles James White.

The Care of the Sick Room. By Elbridge Gerry Cutler.

The Care of the Teeth. By Charles Albert Brackett.

Adenoids and Tonsils. By Algernon Coolidge.

An Adequate Diet. By Percy Goldthwait Stiles.

How to Avoid Infection. By Charles Value Chapin.

Pneumonia. By Frederick Taylor Lord.

New Growths and Cancer. By Simeon Burt Wolbach.

The Causes of Heart Failure. By William Henry Robey.

A Present-Day Conception of Mental Disorders. By Charles Macfie Campbell.

Smallpox and Vaccination. By Benjamin White.

Psychotherapy. By Edward Wyllys Taylor.

HARVARD HEALTH TALKS

LONDON : HUMPHREY MILFORD
OXFORD UNIVERSITY PRESS

HARVARD HEALTH TALKS

SAVING EYESIGHT AFTER MID-LIFE

BY

JOHN HERBERT WAITE, M.D., S.M.
INSTRUCTOR IN OPHTHALMOLOGY
HARVARD MEDICAL SCHOOL

CAMBRIDGE
HARVARD UNIVERSITY PRESS
1927

PRINTED AT THE HARVARD UNIVERSITY PRESS
CAMBRIDGE, MASS., U.S.A.

HARVARD HEALTH TALKS

PRESENTING the substance of some of the public lectures delivered at the Medical School of Harvard University, this series aims to provide in easily accessible form modern and authoritative information on medical subjects of general importance. The following committee, composed of members of the Faculty of Medicine, has editorial supervision of the volumes published:

DAVID LINN EDSALL, A.M., M.D., S.D., Dean of the Faculty of Medicine.

SIMEON BURT WOLBACH, M.D., Shattuck Professor of Pathological Anatomy.

WALTER BRADFORD CANNON, A.M., M.D., S.D., George Higginson Professor of Physiology.

SAVING EYESIGHT AFTER MID-LIFE

SAVING EYESIGHT AFTER MID-LIFE

Importance of Eyesight

IF VISUAL organs had never been developed, it is difficult to understand how species could have evolved beyond the level of the land-grubbing animals which are limited to an atmosphere of sounds and smells. The visual mechanism, brain and eye, reaches its acme in men and birds.

In man, sight is the dominant sense, and it is more informative than all other senses combined. With respect to range, sight is supreme. Taste, touch, and smell function within the range of the immediate environment. Hearing, even with the aid of telephone and radio, is limited to this planet. Sight is the only sense which reaches out to other planets. With

respect to sensitivity, sight is supreme. The eye quickly adapts itself to light intensities covering a range of five billion to one, a span so wide that the eye has no equal either in physical instrument or in sense mechanism. When light-adapted, the eye perceives between two stimuli relatively small differences in intensity. For comparison, touch discriminates difference as small as 1/10, hearing as small as 1/50, and sight with the light-adapted eye as small as 1/200. When dark-adapted, the eye grows very sensitive to dim light. According to Coblentz of the United States Bureau of Standards, the dark-adapted human eye is 300,000 times more sensitive to dim light than is the thermopile, the most sensitive physical instrument.

It is little wonder, then, that man is so dependent upon his eyesight. It informs him about the color and shape of objects. It affords him a means for judging distance. It quickly and thoroughly ex-

plores the environment before he moves into it, and it gives him immediate warning of danger. It guides the hand in industry, and the foot in traffic. Through the visualization of written symbols, it gives man the key to all recorded literature. Without eyesight, modern life is fraught with a thousand hazards, and mental development is confronted with a thousand obstacles.

Of the 100,000 blind people in the United States, 45 per cent have been blinded from causes external to the body, and 55 per cent have been blinded from causes within the body. Noteworthy efforts are being applied to control the external causes, such as "sore eyes of babies" which has contributed 2 per cent, trachoma which has contributed 3 per cent, and industrial accidents which have contributed 13 per cent of the total blindness. These figures from the 1910 census are from an analysis by Best of 29,242 cases in which the cause of blind-

ness was known. It is to be noted that external causes produce blindness mostly before the fortieth year, and with decreasing frequency thereafter.

Fifty-five per cent of the total blindness results from causes within the body, which operate scarcely at all before the fortieth year of life, but with increasing frequency as age advances. That the eyes do wear out before the body wears out in many of us is shown by the following table:

Age Group	Number Blind per million in this age group
1– 4	57
10–14	219
20–24	243
30–34	329
40–44	532
50–54	961
60–64	1,977
70–74	4,589
80–84	12,833

This is not because blind people outlive those who see, but because degenerative

disease after mid-life attacks the eyes more and more frequently with advancing age. For example, according to the figures of Magnus, in the age period 35–44 years, glaucoma causes 11 per cent of all blindness found in that age group, 27 per cent of that in the group 45–54 years, and 58 per cent of that in the group 55–64 years.

The Problem: Conservation of Eyesight after Mid-Life

In spite of the fact that much of it is preventable, practically no organized effort has been made to curb the blindness from degenerative disease which comes after the fortieth year. While it is not possible with the present state of knowledge to prevent the degenerations that come with age, it has been repeatedly shown possible to prevent the damage to sight through early recognition and early treatment, notably in glaucoma which leads all other causes of blindness after

mid-life. In all degenerative disease affecting sight, there are two important considerations. First, efficient early treatment is fundamental because a loss of one-half of the normal sharpness of vision will practically always unfit a craftsman for his accustomed work. (A visual acuity of 20/100 represents a loss of 51 per cent; Snell and Stirling, *Archives of Ophthalmology*, 1925, page 443.) Second, early treatment is not possible until the disease is recognized, and early recognition of degenerative disease by the lay patient is extremely difficult because of its insidious, painless onset.

The Normal Visual Mechanism

In order to understand better how eyesight may be conserved, let us review the essential structure and function of the visual mechanism.

The posterior surface of the brain receives nerve impulses sent to it from the eye, and interprets them in terms of

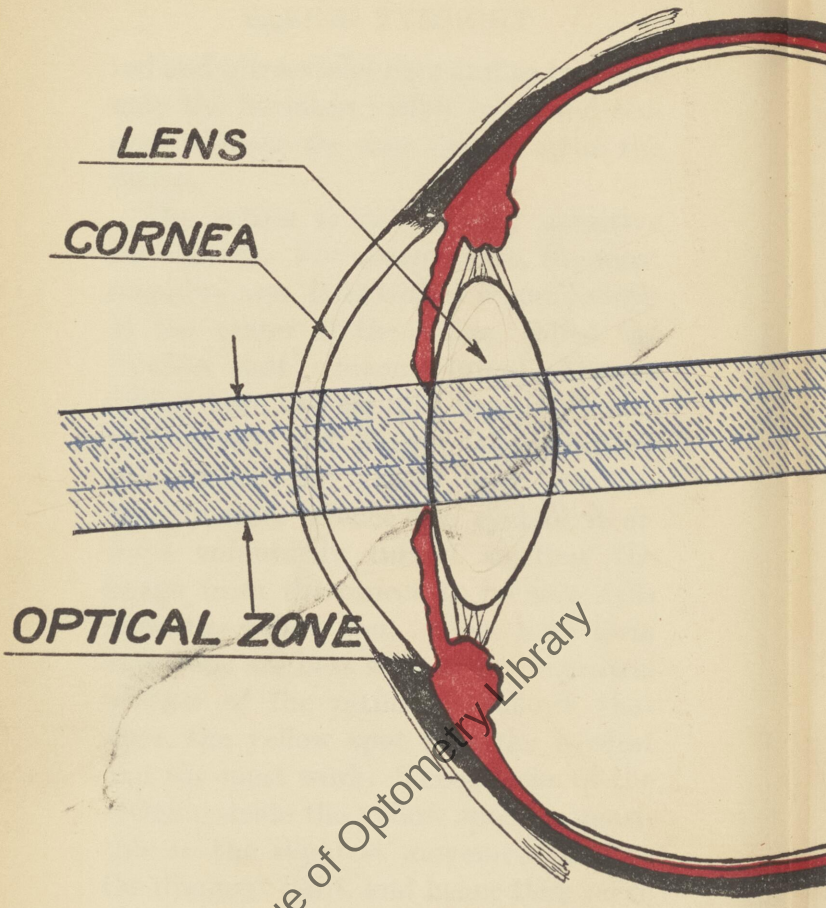
color, form, and depth. An injury here destroys sight as surely as does an injury to the eyeball. Shielded by the bony skull, the brain is protected against external injuries much more than it is against those from within the body, particularly from degenerative processes.

The receiving station for light stimuli is to be found in the light-sensitive tissue of the eye, called the retina, where the physical energy of light is converted into nerve impulses which pass to the brain. In fact, the retina is an outgrowth from the brain, and it remains an integral part of it. Since neither brain alone nor eye alone can see, both should be considered as a unit, functionally and anatomically.

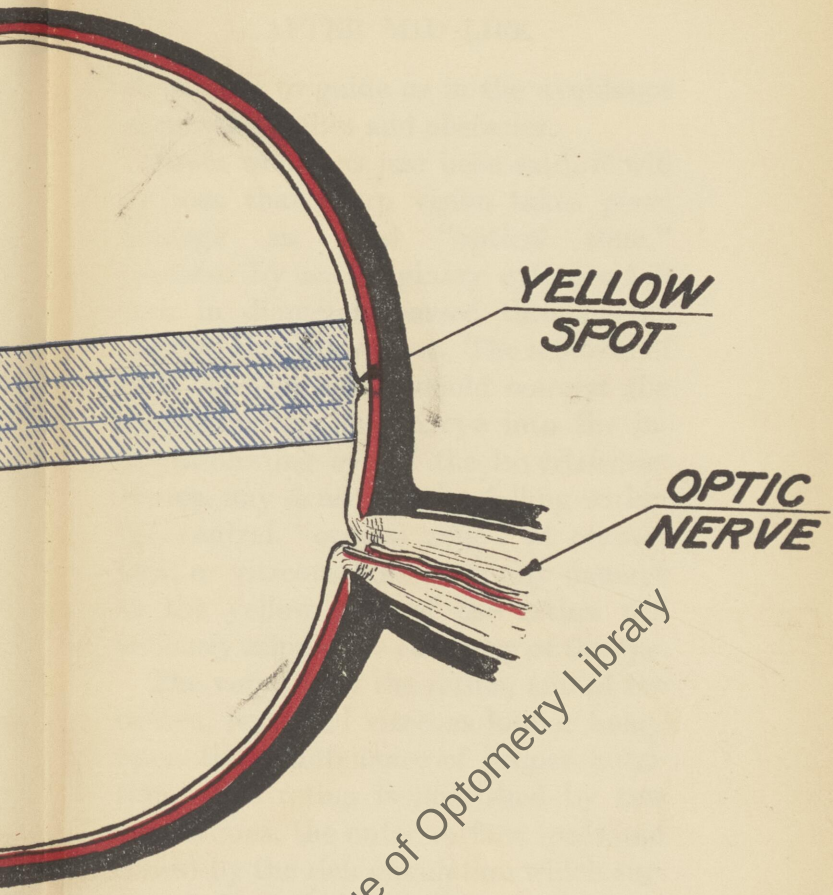
Before reaching the retina, all rays of light must pass through the transparent media of the eyeball, namely the cornea, the crystalline lens, and the vitreous humor. These media have a double function; first, to filter out all harmful infra-

red and ultra-violet rays and to transmit only the harmless visible rays; and second, to focus the rays sharply upon the retina.

The retina is not equally sensitive throughout. For sharp vision, the most sensitive area falls within a small circle at the center of the retina, called the "yellow spot" (macula lutea). Images falling here are sharply seen if properly focussed, and images falling farther away from the yellow spot are less sharply seen. Hence the eyeball is always voluntarily turned so that the image from the object to be seen falls upon the yellow spot. Since both form and color are best seen with the central portion of the retina, it follows that upon the yellow spot falls the hardest and the most work. Those parts of the retina outside the yellow spot are sensitive to the slightest movement and to the dimmest light, and hence they serve to orient us with respect to our environ-



Grateful acknowledgment is gladly made to Mr. E.
which illustrate the te



Mr. E. B. Kirk for the excellent drawings
te the text.

ment, and to guide us in the avoidance of moving bodies and obstacles.

From what has just been said, it will be seen that sharp vision takes place through an axial "optical zone," bounded by an imaginary cylinder 1/6 inch in diameter, passed through the pupil to the yellow spot. The removal of this "optical zone" would convert the discriminating human eye into the indiscriminating eye of the invertebrate. Hence, any dense opacity falling within the central "optical zone" of cornea, lens, or vitreous humor, or any damage to the yellow spot of the retina will seriously cripple the efficiency of the eye.

The very life of the retina, and of the cornea, lens, and vitreous humor hangs upon the maintenance of proper nutrition. The retina is nourished by two circulations, the outer surface (rods and cones) by the rich circulation which surrounds it, and the inner surface (nerve fiber layer) by the retinal vessels which

spread out over it. The cornea is nourished by lymph poured out from the loops of blood vessels adjoining it. The lens and vitreous humor are bathed and nourished by the aqueous humor, which emerges through the pupil, and escapes at the angle of the anterior chamber. Hence aqueous humor is constantly entering and leaving the eyeball, at the rate of one drop every ten minutes, and its flow is maintained under a pressure equivalent to 10 to 25 millimeters of mercury. This pressure tends to keep the coats of the eyeball tautly spherical, and thus to promote optical efficiency. If the pressure falls appreciably below normal, the coats of the eyeball become wrinkled, and hence unfit for optical purposes. If the pressure rises above normal, the contents of the eyeball are subjected to the full force of the increased pressure against the unyielding sclera. The condition of high pressure

with all of its serious consequences is known as glaucoma.

Glaucoma

Glaucoma presents a spectacle which has no parallel in modern medicine. On the one hand, it is the most prolific single cause of blindness in the United States, causing 1/3 of all blindness arising after the fortieth year. On the other hand, it practically always yields to certain forms of treatment, established upon fifty years of successful experience, provided the patient receives the treatment early enough. In spite of this combination of facts, glaucoma is being allowed to run its destructive course unopposed, and to leave in its wake thousands of people needlessly blind.

The popular idea of glaucoma is that of an acute, painful affliction, which rapidly progresses toward blindness. If all glaucoma were of the acute type, each victim would be compelled by his own

suffering to seek relief while his sight could still be saved. As a matter of fact, nearly all the glaucoma one sees is of the chronic type, which gives no warning of its presence sufficient to attract the attention of the patient. It attacks both eyes painlessly, but usually one eye before the other. It frequently spares sharp central vision of the yellow spot until last, allowing the patient to read fine print, when the rest of the retina is hopelessly blind. Finally, after the patient finds himself blind in one eye, is he persuaded to seek an ophthalmologist for the first time. There, he is told that he is incurably blind in one eye, and often that his other eye is seriously affected.

Here, then, is the key to the problem. Glaucoma is subtle, because it gives no outspoken symptoms. If all people past 40 could be brought to realize that certain insignificant symptoms are in reality the first warnings of serious trouble, and if they could be persuaded that the

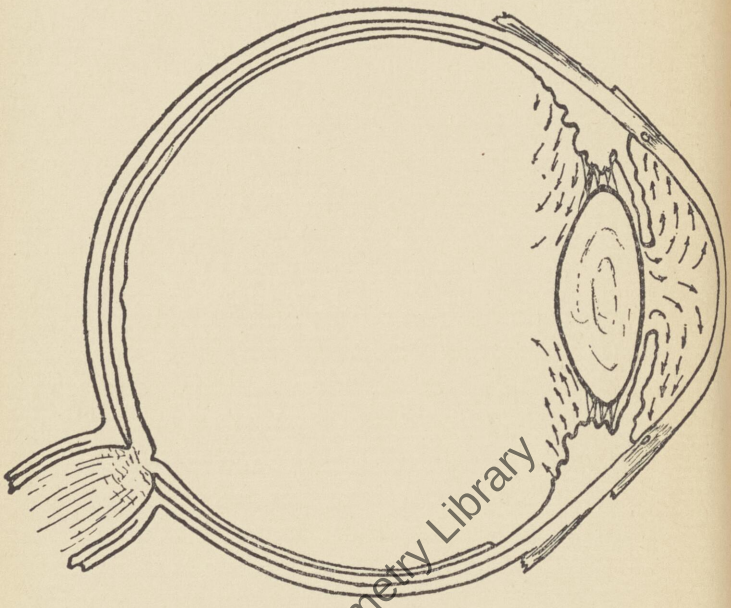
most expert advice is none too good in glaucoma, then a large amount of blindness from this cause could be prevented.

The cause, the symptoms, and the treatment of glaucoma all center around a constant or a periodic increase in intraocular pressure.

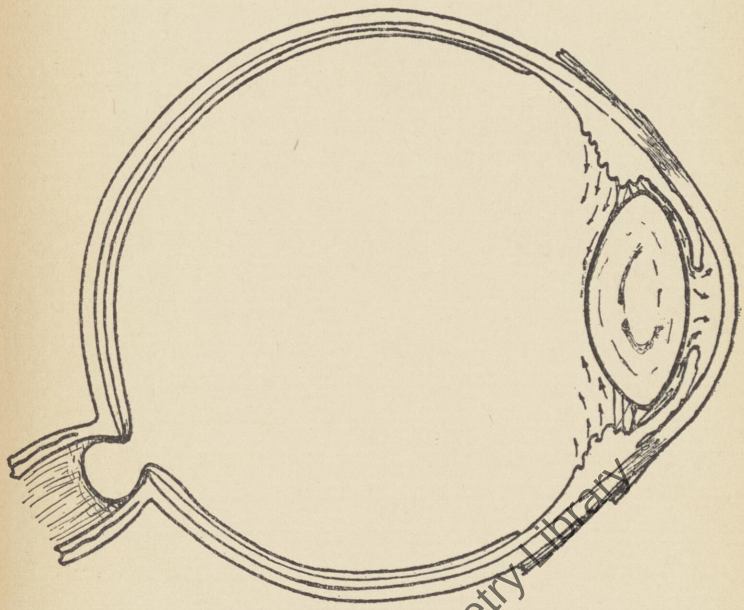
If one injects certain harmless dyes into animals, one can observe the presence of the dye in the aqueous humor after some minutes, and one can follow the circulation currents in this humor as it pours forward through the pupil. Further, if one injects India ink into the vitreous humor of an animal's eye, one can find later on innumerable carbon particles in the tissue meshes of the filtration angle. Since the pressure within the eyeball is balanced at a constant level, the rate of outflow of aqueous humor from the eye must equal the rate of inflow through the ciliary processes. From this it follows that increased pressure may arise either from too rapid an inflow

or from too slow an escape of fluid from the eye. The second theory, the retention theory, finds the most general acceptance.

It is well known that a marked shallowing of the anterior chamber nearly always precedes an attack of primary glaucoma. Some attribute this shallowing to a swelling of the ciliary processes, others to an increase in the size of the lens, and still others to an increase in the volume of the vitreous humor. It is not impossible that all three factors contribute to the common effect. At any rate, whenever the anterior surface of the iris is pressed against the posterior surface of the cornea, there is at once a mechanical obstacle to the free escape of aqueous humor, and the pressure will rise. The pressure will stay up so long as the iris and the cornea are in contact. It has been observed that the iris and cornea grow together when the contact is prolonged, and in this permanent barrier



NORMAL EYE. Note the appearance of the optic nerve head, and the depth of the anterior chamber. Arrows show the origin and currents of the circulating aqueous humor, which escapes from the eyeball where the root of the iris meets the cornea, the so-called "filtration angle."



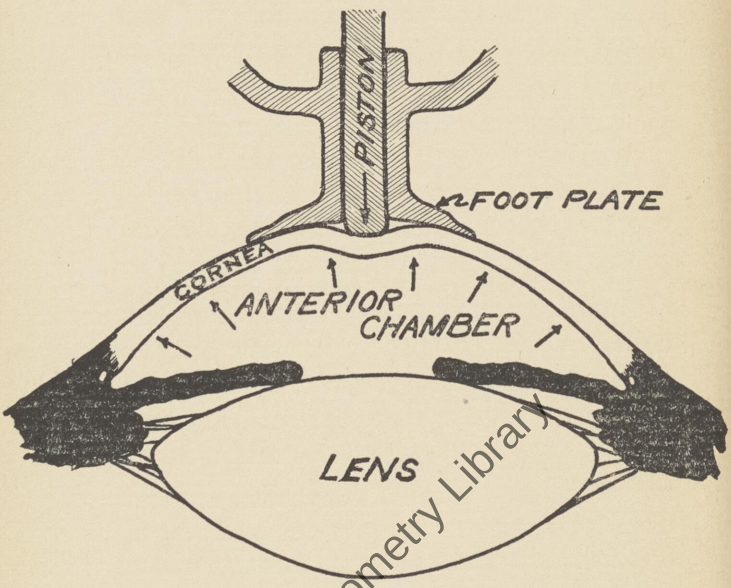
GLAUCOMATOUS ADULT EYE. Note that the iris is pressed against the cornea blocking the free escape of aqueous humor, and producing increased pressure within the eyeball. All coats of the eyeball withstand pressure better than the optic nerve head, which is soon hollowed out and killed unless the pressure is reduced to normal.

glaucoma finds its origin. A permanent barrier of this kind is more likely to occur in the small, far-sighted eye, rather than in the large, near-sighted eye. The hereditary element in glaucoma may be partly explained on this basis.

Elliot likens the filtration angle in glaucoma to the "cockpit" where fighting takes place, and the optic nerve head to the "graveyard" where the killed nerve fibers are forever dead. In certain rare instances, where increased pressure is present in a baby's eye, the elastic coats of the eyeball are ballooned out to two or three times their normal size. On account of its enormous size, such an eye is called "ox-eye" (buphthalmos). Increased pressure is not compensated for in this fashion in the adult eye, where the coats are inelastic. Unfortunately, the most vital spot in the adult coats, being where the optic nerve escapes, is also the weakest spot and hence the most vulnerable. Increased pressure within the

adult eye exerts its full force against the optic nerve head, balloons it out in the form of a cup, and squeezes, stretches, and kills the delicate optic nerve fibers. There is no possibility of regeneration of optic nerve fibers after they are killed, and hence sight is irrevocably lost.

The signs and symptoms of glaucoma are directly or indirectly the consequence of increased intraocular pressure, and they may roughly be gauged by the amount of increase. In the more chronic types of glaucoma, the increase of pressure may be very slight, or it may be present only during a few hours of each twenty-four hour period. Here symptoms are conspicuous by their absence. In glaucoma, where the pressure is sustained and higher, the symptoms are more marked, and the damage to sight is more rapid. Finally, in the more rare, acute, fulminating glaucoma, the pressure may be very high, and an erstwhile



PRINCIPLE OF TONOMETRY

seeing eye may run through agonizing symptoms to complete blindness within twenty-four hours. Let us enumerate the signs and symptoms according to anatomical structures which give rise to them.

1. *Pressure.* — Just as one can indent a tennis ball with the finger tips, so can one indent an eyeball having normal pressure. With educated finger tips, one can learn to estimate the pressure roughly. More accurate measurement is possible through the use of an instrument called the tonometer, which measures the state of tension of the cornea. Eyes are just like people, some can stand a lot of punishment, and others can stand but little. What might be a perfectly normal tension for a resistant eye, may be entirely too high for a less resistant eye. Hence there is no such thing as a "normal" tension, applicable to all eyes. Each eye has its own normal, and this

may range anywhere between an equivalent of 10 to 25 millimeters of mercury.

2. *Cornea.* — The higher the pressure, the more disturbance is there in the lymph-flow from the cornea. The surface tissues of the cornea are stippled with fine droplets of retained fluid, giving a bedewed appearance, and disturbed function. The patient may complain that he "seems to be looking through smoke," or that he "sees colored rings around bright lights." One gets these same reactions upon looking through a window pane which has just been breathed upon. Neither smoky vision nor halos are characteristic of glaucoma, because they may be present in any condition which gives rise to accumulation of fluid in the cornea.

3. *Pupil and Iris.* — Increased intraocular pressure blocks the impulses which traverse the nerves running forward between the coats of the eyeball to

regulate the size of the pupil. The higher the pressure, the more dilated the pupil, and the more sluggish is the action of the iris. The release of high pressure allows the pupil to become normal again.

4. *Focussing Muscle* (Ciliary Muscle). — Variations in pressure cause variations in the power of the eye to adapt itself to a reading focus. One of the commonest early symptoms of glaucoma is a difficulty in reading, even after a reading glass suitable for age has been carefully fitted. Any eye older than 45 years is likely to have focussing difficulties, which reside in the ageing lens, but which can always be corrected by the use of a proper reading glass.

5. *Lens.* — The lens usually remains transparent until late in the disease, when an interference with its nourishment may cause a progressive cataract. In some forms of glaucoma, the lens may assume a greenish hue, from which char-

acteristic the name glaucoma has been derived (γλαυκός = sea-green). After cataract has developed, the disease is frequently miscalled "cataract" and the fatal mistake is made of waiting until the "cataract" ripens before instituting treatment. Any delay in glaucoma allows blindness to progress.

6. *Cupping of the nerve head* has already been alluded to. On account of its importance, we may refer to it again. Cupping may be seen in the living eye only by looking into the eye with an ophthalmoscope. Under a magnification of 15 diameters, the normal nerve head appears to be on the same level with the rest of the structures seen, and the retinal blood vessels are conspicuous as they emerge from the substance of the disc. In early glaucoma, it may be impossible to find more than a slight depression of the disc tissues, but as glaucoma advances, finally the entire disc is

ballooned away from the observer. It gives one the impression of looking down into a bowl, seeing only the mouth of the bowl and its floor, but not being able to see the receding sides. The retinal blood vessels bend sharply over the mouth of the bowl, and disappear from view, only to reappear upon the floor of the bowl. The optic nerve fibers cannot be seen because they are transparent, but they also bend sharply over the edge of the bowl, where they are squeezed, stretched, and killed. Whenever found in the fully developed form, the glaucoma cup affords an extremely important aid for a positive diagnosis. As death of the nerve fibers takes place, the disc loses its normal pink color, and becomes a pale gray, or green-gray.

7. *The Retina.* — With the onset of increased pressure, there is an obstruction to the free escape of blood from the eye, and both the retina and the vascu-

lar coat show congestion. For perfect function, the retina requires a free circulation. With a sluggish circulation, the rods and cones, and the ganglion cells are not sufficiently nourished; they are poisoned in their own waste products, and they become torpid. The patient may notice, if he is observant, that he requires more light to read by, and that he cannot become accustomed to dim light in a darkened room as readily as he once could. As the disease advances, this defect is apt to increase. We have at the Massachusetts Eye and Ear Infirmary an instrument with which a careful measurement of the "light sense" can be made, and we have found in glaucoma eyes a constant interference with the "light sense," at first slight, later more and more marked.

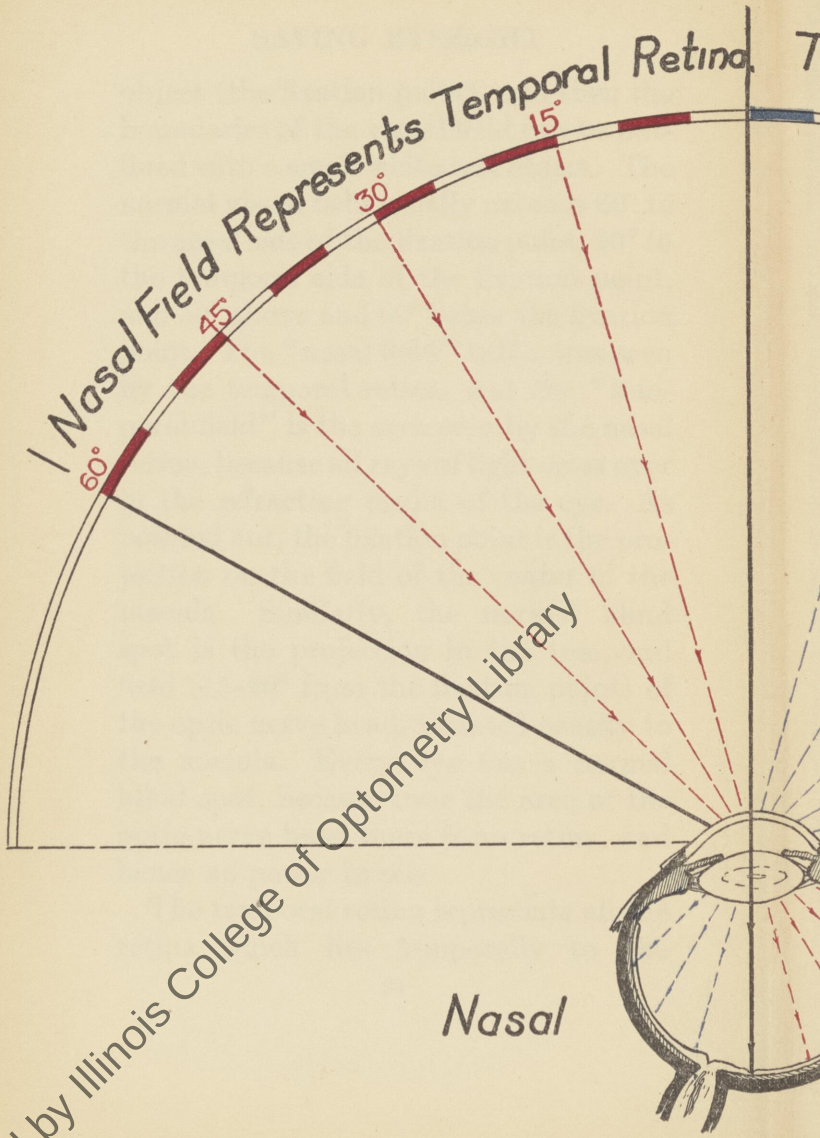
8. *Visual Fields.* — A careful record of the visual field gives more accurate information about the progress of glau-

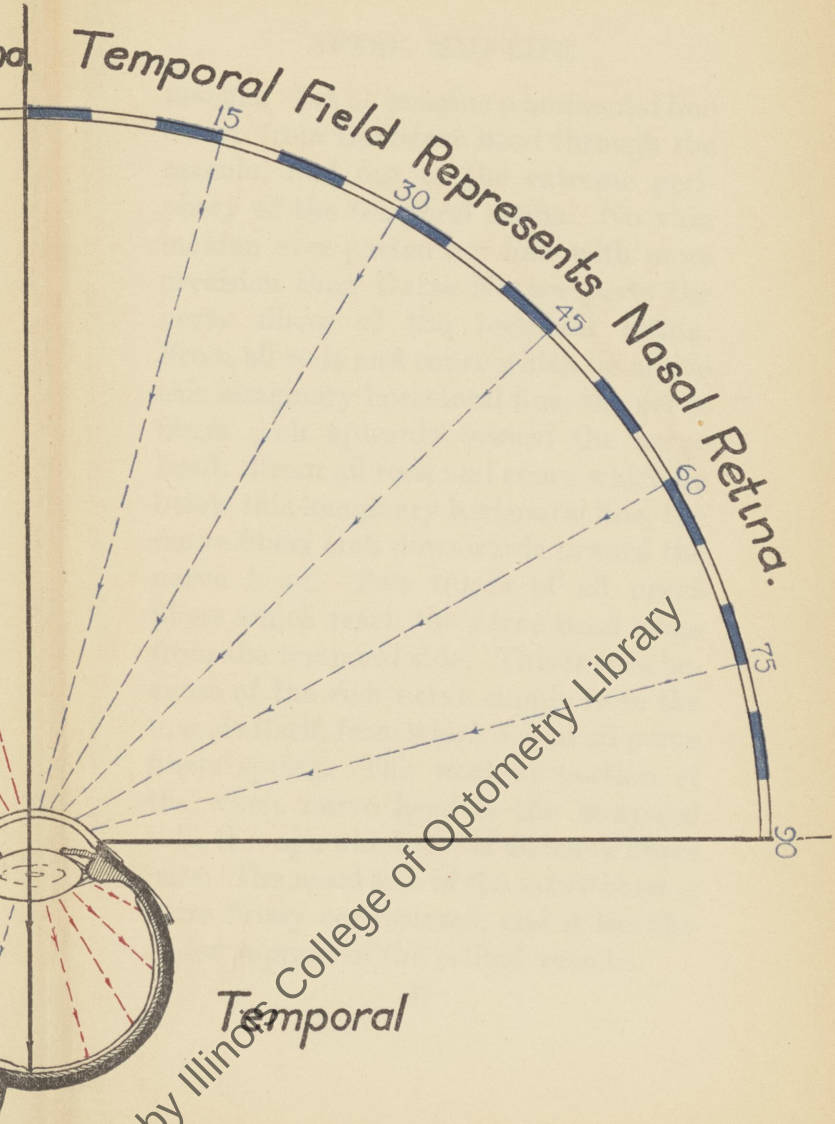
coma from beginning to end, than any other single test. Increased intraocular pressure, cupping of the nerve head, and changes in the visual field that are characteristic of glaucoma, together form a diagnostic triad upon which an unfailing diagnosis can be made. Diminished light sense, variable focussing power, and colored halos strongly arouse our suspicions of glaucoma, but they do not clinch the diagnosis.

The normal visual field represents the extent of the environment which can be perceived by the eye held in a fixed position. In other words, if we look straight ahead, we are at the same time conscious of objects to the right of us, objects to the left of us, and objects above and below the level of our eyes. Since in man, the visual field of the right eye overlaps the visual field of the left eye, each eye must be tested separately. With one eye blindfolded, the other eye looks at an

object (the fixation point), and then the boundaries of the visual field can be outlined with a small white test object. The normal visual field usually extends 60° to the nasal side of the fixation point, 90° to the temporal side of the fixation point, and 50° above and 60° below the fixation point. The "nasal field" is the area seen by the temporal retina, and the "temporal field" is the area seen by the nasal retina, because all rays of light cross over in the refracting media of the eye. As pointed out, the fixation point is the projection on the field of the center of the macula. Similarly, the normal blind spot is the projection in the temporal field (15–20° from the fixation point) of the optic nerve head, situated nasally to the macula. Every eye has a normal blind spot, because over the area of the optic nerve head there is no retina, and hence no power to see.

The temporal retina represents all the retina which lies temporally to the

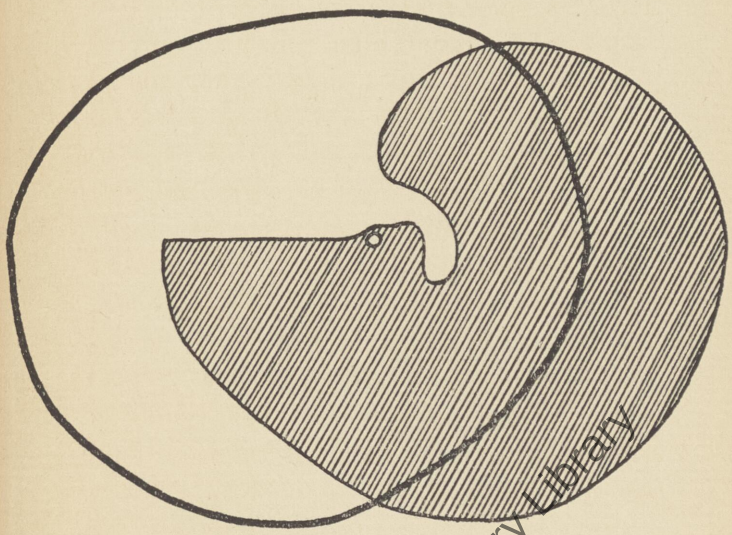




macula. Let us imagine a horizontal line drawn from the nerve head through the macula, and out to the extreme periphery of the temporal retina. No vain maiden ever parted her hair with more precision than Dame Nature parts the nerve fibers of the temporal retina. From all rods and cones which lie above this imaginary horizontal line, the nerve fibers arch upwards toward the nerve head. From all rods and cones which lie below this imaginary horizontal line, the nerve fibers arch downwards toward the nerve head. Two thirds of all nerve fibers which reach the nerve head come from the temporal side. This is true because of the rich nerve supply from the macula itself, from which 1/3 of all nerve fibers spring. The weakest portion of the whole nerve head is the temporal side, through which 2/3 of all nerve fibers pass. The nasal side of the nerve head is more firmly constructed, and it has the added support of the retinal vessels.

When pressure comes, the first part of the nerve head to give way is therefore the temporal or weaker side. Owing to the curious anatomical distribution of nerve fibers, arching from the temporal retina, it will be seen that the first visual field defects in glaucoma are located in the "nasal field" and have characteristic outlines. In the order of their appearance, there is first a vertical enlargement of the normal blind spot up or down, second a peripheral defect in the nasal field up or down and called the "nasal step," third an arcuate enlargement of the blind spot to meet the nasal step and called Bjerrum's scotoma, and fourth a concentric contraction of the entire peripheral field.

Since the nasal fields overlap, it will become evident at once that the defect in one eye is covered up by the integrity of the other. Unless the good eye is covered, and unless a most careful search is made in the nasal field of the affected



SHADED FIELD — from glaucomatous right eye.
UNSHADED FIELD — from normal left eye.

Since the nasal portions of the fields overlap, the typical nasal defect in glaucoma is wholly obscured when both eyes are open.

The circle (○) in center of plate represents the fixation point.

eye, the defect is not likely to be found. Since an examination of this kind is never made by the patient himself, early defects in the visual field are never found by patients.

More important than any of these considerations is the fact that acute central vision is almost always spared until very late in the disease. Enough of the nerve fibers leading from the yellow spot are preserved to allow the patient to read ordinary newspaper print, even in well-advanced glaucoma. On this account, the patient is falsely fortified by a feeling of security. Finally, the terrible drama closes with the snuffing out of the last spark of sight.

Treatment for Glaucoma

The only bright spot in the blackness of glaucoma is the fact that measures have been discovered which will control the pressure, and therefore will prevent or at least delay the onset of blindness. While

it is impossible to prevent the disease itself, it has been repeatedly shown that it is possible to recognize glaucoma fairly early, and to prevent or at least to delay its damage through controlling the pressure.

Laqueur in 1876 first observed that certain drugs which strongly contract the pupil have the virtue of reducing the pressure in a glaucomatous eye. The effect of the drug lasts only for a few hours, and hence for prolonged action it must be instilled several times every day. The ocular tissues slowly develop an immunity to the drug, and so the strength of the drug must be constantly increased. Finally, the maximum strengths fail to achieve the desired result, and the drug no longer holds the pressure within safe limits. In brief, the drug method of controlling glaucoma is at best only a temporizing method, and its exclusive use is justified only in people so old that their

expectation of life is less than the period of effective action of the drug.

De Wecker has said, "If miotic drugs have never cured a case of glaucoma, they have prevented many glaucoma patients from being cured." The point to his remark is that the tissues of the glaucomatous eye rapidly undergo degenerations, which make later surgery hazardous.

Another temporary measure is the use of gentle massage, which forces fluid out of the eye, and affords a brief interval of freedom from abnormal pressure.

If one's house drain is obstructed, one has two alternatives. One may temporize by pouring alkali or hot water down the drain, in the hope that drainage may thereby become reëstablished. In the long run, such a method is bound to fail. On the other hand, one may take the radical step, and send for the plumber to open the drain. The earlier the radical step is taken in glaucoma, the

more likely is there to follow a complete and satisfactory relief from pressure, and hence from damage.

All surgical procedures in glaucoma aim to reopen old drainage channels, or to establish new ones. In 1851 von Graefe discovered that the removal of a wide piece of iris down to its root could reopen sufficient drainage channels to control the pressure. Thousands of eyes in acute glaucoma have been completely and permanently cured by this procedure. No surgical triumph has ever been more brilliant in its beneficial results than von Graefe's iridectomy. Later surgical procedures have been devised to create new channels for the escape of aqueous humor, and many of these produce satisfactory results. With every operation, there is a certain amount of risk to eyesight, the risk being much less when the operation is performed while the tissues are healthy. Without operation, the ultimate result of glaucoma is

blindness. With successful operation comes permanent relief. The goal is worth the risk!

Other Causes of Blindness after Mid-Life

While glaucoma is the chief problem in the conservation of eyesight after mid-life, there are other important and difficult problems. Blindness may arise in more ways than one, from degenerations or new growths in either brain, optic nerve, or eyeball. It may come from deterioration of the visual areas within the brain. It may come, as in glaucoma, from death of the optic nerve fibers where they escape from the eyeball. It may follow death of the retina itself from obliterative processes within the arteries and veins which supply the retina with blood. With brain, optic nerve, and retina in perfect health, practical blindness may result from dense

opacity in the optical zone of the cornea or lens.

Degenerated Blood Vessels of the Eye and Brain

The tissues of the heart and blood vessels are called upon to work continuously from birth until death, and they are often the first to suffer degenerations. When the circulatory system starts to fail, degenerations in other tissues begin. Every man is said to be "just as old as his arteries."

The arteries to the eye are but the terminal branches of the arteries to the brain, and hence they suggest the state of preservation of the latter. With an instrument, called an ophthalmoscope, one can see and study the naked arteries and veins in the retina under a magnification of fifteen diameters. With advancing age, nearly everyone shows certain changes in the retinal vessels, which however do not interfere with

sharp vision. In certain people, these changes reach a grade sufficient to cause disturbance, even total loss, of vision. Fortunately, the damage is usually less in one eye than in the other. The changes consist of gradual or sudden obliteration of the vessel, with or without associated hemorrhages, attended by death of the retina from which nourishment is cut off. Changes of this kind are sometimes followed by secondary glaucoma, or by cataract formation.

The presence of degenerated blood vessels may indicate bad heredity, bad living, or unusually severe stress and strain, and once established it does not yield to any immediate treatment. Since diseased vessels in the eye usually signify diseased vessels in the brain, the best program for the patient to follow is a relaxation from mental and physical strain, in order to forestall the ever-impending vascular accident in brain or eye.

Cataract

Cataract, the progressive opacification of the lens, may be the first sign to the patient or the physician of serious disease within the eye, and hence its cause should always be carefully investigated. Senile cataract is the degeneration of the lens alone, the other structures of the eye remaining normal. Complicated cataract is the degeneration of the lens which follows glaucoma, new growth, vascular changes, or inflammation within the eye.

Cataract may arise following diabetes, following the ingestion of poisons such as ergot or naphthalin, or following a blow upon the eye.

The early symptoms of senile cataract depend upon the location and the density of the opacity. If it is placed out of the optical zone, there are no noteworthy symptoms. If placed within the optical zone, it may cast a shadow upon the

retina which is interpreted as a floating speck, or it may refract certain rays of light more strongly than does the clear lens substance surrounding it, thus giving the patient the sensation of double or triple vision with that eye. If the opacity is small, and lodged within the optical zone just behind the pupil, the patient may see poorly in bright light, and better in dim light, because with the dilation of the pupil he can see around the opacity. Vision fails as the opacity increases, until finally the patient cannot read print. Since no cataract is dense enough to exclude light, the patient should be able to see light and to tell accurately from which direction it is coming, if the eye is otherwise healthy. "Ripening," the progression of the opacity, proceeds at different rates in different individuals, and the rate cannot be foretold. While there is no known way of arresting, or of clearing up a cataract once it has started to form,

there is a positive cure in the surgical removal of the lens.

The early symptoms of complicated cataract embrace all the early symptoms of every different condition which gives rise to this form of cataract, and it is impossible here to consider them all in detail. The removal of a complicated cataract by surgery frequently offers no improvement in vision for the reason that the retina is diseased and cannot function. In complicated cataract, the careful search for the cause will not infrequently yield information which can be applied to promote the general or the ocular health of the patient.

Melanotic Sarcoma of the Eye

Finally, there is a condition arising most commonly between the fortieth and sixtieth years of life, which menaces not only sight, but even life itself. This condition is melano-sarcoma, a type of "cancer" which starts in the middle coat

of the eyeball. Its beginning is painless, and it does not affect the sharpness of vision unless located at the yellow spot. It may release some of its malignant cells through the blood stream to other parts of the body, even in its early stages. Once the tumor cells spread beyond the eye there is no hope of saving the life of the patient.

As the tumor grows, it begins to affect vision, either by obstructing the optical zone, or by causing a separation of the retina. Complicated cataract, or secondary glaucoma may develop at any time. Finally, the tumor may break through the coats of the eyeball, and extend into the surrounding tissues.

There is no way to prevent the occurrence of this or any other new growth. The one fatal mistake is not to recognize the presence of the tumor at an early stage, and not to remove the eye at once. To call the condition "cataract" or "glaucoma" favors delay in removing

the eye, and allows the tumor cells to spread from the eye to other parts of the body.

Summary

To save eyesight after mid-life, remember

1. That periodic examination by an oculist is necessary, because of the insidious onset of degenerative disease of the eyes.
2. That cataract may be part of a process, not limited to the crystalline lens, but heralding the presence of a more deep-seated and serious disease of the eye.
3. That blindness from glaucoma, which at present amounts to one-third of all blindness arising after mid-life, can usually be held in check if the patient will only cooperate with his oculist.

Date Due			